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PRIMARY GLAUCOMA*
Its Etiology, Symptoms, Diagnosis

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Wilmington, Del.

Glaucoma is a destructive disease. It is of frequent occurrence. de Grosz of Budapest states that more blindness results from glaucoma than from syphilis, tuberculosis, gonorrhea or even injuries. It has been stated that of all blindness in the United States 4.6 to 6 per cent is caused by glaucoma. More accurate accounting of data in recent years, according to the executive director of the National Society for the Prevention of Blindness, indicates that 11 per cent is caused by glaucoma. Since the major portion of this paper was written, originally prepared for and presented before ophthalmologists, Harry Gradle has written an editorial in the American Journal of Ophthalmology in which he states that of all the blindness in the United States between 15 and 20 per cent is due to glaucoma, and that this fact is not entirely comprehended by the ophthalmologists of the country. The disease is not diagnosed, first, because non-medical refractionists practically never diagnose preglaucomatous cases; second, because of incompetent medical men practicing ophthalmology; third, because of neglect by the patient of failing vision in one eye; fourth, because family physicians are not as cognizant of glaucoma as they should be. Blake of New Haven calls glaucoma "cancer of ophthalmology"—not, of course, cancer of the eyes. It is comparable to cancer in that we do not know its cause, we do not know when it begins, and the same question, "Can you cure it?" may be asked. General practitioners and the lay public should be familiar with the cardinal symptoms of glaucoma so that patients may present themselves for diagnosis early enough to arrest the disease. The marked decline of blindness from ophthalmia neonatorum over the past 35 years should make us think that through cooperation of the members of our profession a much lower percentage in blindness from glaucoma may be attained.

What can we say about the causes of glaucoma? The condition is usually called hardening of the eye. Hippocrates recognized hypertension but it was not until after von Helmholtz invented an ophthalmoscope that cupping of the disc was recognized. The accepted view by ophthalmologists is that glaucoma is a disease characterized by increased tension of the eyeball, that this is produced by a narrowing or closure of the pathways of exit of the fluids from the anterior part of the eye, and that, failing reduction of this tension by the use of miotics (and more especially pilocarpine), an operative procedure relieving this tension is the only means of relief. Magitot of France and a growing number of writers, Frank Park Lewis points out, are accepting the view that the closure of the anterior pathways is due to the increase in the amount of the intraocular fluids and is a consequence rather than a cause of the disastrous series of events accompanying the disease. But what increases the amount of the intraocular fluids?

Schmelser[1] published some studies he made on the intermediate metabolism of patients with and without glaucoma. He found a large majority of patients with glaucoma had a pronounced hypercholesteremia and many gave a positive xanthoproteric reaction and impairment of hepatic function. He tested 78 with glaucoma and 70 without. Impairment existed from the fact that during tolerance tests with amino acids (aminoacetic acid) the deaminization was retarded in contradistinction to controls of the same age. He thinks that hepatic disturbances play an important part in glaucoma.

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(1) Archiv fur Ophth. (Dec., 1939)

Luedde[2] has called attention to the probable function of the capillary epithelium of the small blood vessels in the eye, and the probable escape of fluid through the permeability of the peripheral cornea, as a factor in glaucoma. He calls attention to the report of Kirwan on the outbreak of epidemic dropsy in Calcutta, 1934-35, during which time Kirwan saw in his hospital 1,695 cases of primary glaucoma in association with this disease. Very high tension occurred in both eyes. There was no lowering of the tension by miotics, normal or deep anterior chambers were present, there was absence of any inflammations or external signs of congestion, and the condition occurred in persons irrespective of age. The toxemia is apparently caused by parboiled rice, as patients quickly recover after the stoppage of rice in earlier cases, and relapses occurred when the rice was resumed.

Barkan believes that the production of blockage of the sclerocorneal trabecula by formation of pigment or the migration of iris pigment is an important cause. This seems to be borne out in the case of Negroes, who have lots of pigment and in whom glaucoma is very destructive, very frequently resulting in blindness in spite of anything that is done. With a prismatic contact glass and a corneal microscope the angle of the anterior chamber can be inspected. This procedure is known as gonioscopy of Troncoso. If the angle is open and access to the walls of Schlemm's canal is free, miotics reduce tension through neurogenic or vascular action, Barkan claims, rather than mechanically by changing the configuration of the angle. The majority of cases studied have shown an open angle. The type in which the angle is narrowed is benefited mechanically by miotics pulling the iris away from the entrance to the angle. Studies of this angle have shown that adhesions are probably the cause of obstruction of the flow of fluid rather than obstruction of Schlemm's canal being the cause of the adhesions.

The condition of glaucoma is not all due to a single factor. The activities of all organs are controlled and maintained by a regulatory mechanism in which the main factors appear to be chemical, neurogenic and hormonal (Duke-Elder). Duke-Elder and others think that primary glaucoma apparently is not a local disease but seems to be a local manifestation of some general condition not yet known.

(2) Amer. Jour. Ophth. (April, 1940)

CLASSIFICATION OF GLAUCOMA

1. Primary Glaucoma:
   a. Acute inflammatory, or acute congestive, or acute incompensated.
   b. Chronic inflammatory, or chronic incompensated.
   c. Chronic simple, or chronic noninflammatory, or compensated.
2. Infantile (hydrophthalmus or buphthalmus).
3. Secondary—to acute iritis, uveitis, choroiditis, cataract, injuries to lens, tumors, thrombosis of central vein, glaucoma capsulare (secondary to exfoliation of the lenticular capsule), etc.
4. Absolute.

This paper, as stated in the heading, is confined to the discussion of primary glaucoma, and primary glaucoma means a glaucoma the cause of which is not yet known.

ACUTE INFLAMMATORY GLAUCOMA

Acute inflammatory or acute incompensated glaucoma occurs usually in one eye, more frequently in women than in men (about three to two), and in older people (38 to 70 years), and more in hyperopes. It is usually seen in the thin and wiry individual. It sometimes occurs in families.

We find in textbooks three factors as the probable etiology in the production of the clinical picture of this form of glaucoma:

(a) The anatomical construction of the eye, as in the small eye of hyperopia with an increased angular acuity of the anterior chamber.

(b) The interruption of the pressure regulating mechanism.

(c) A temporary rise in systemic blood pressure.

The choroidal vessels are under the control of the sympathetic nervous system. When this is out the vessels are filled, and the choroidal space can be increased four times in size. This may be done by a big meal, alcohol, anger, worry. The sympathetic nervous system can be stimulated with adrenalin, which

will contract the vessels. Under normal conditions the sympathetic system takes care of the blood supply and causes contraction and emptying of the vessels.

Symptoms

Usually there are prodromal symptoms lasting for a short time (from one to three hours) and several months apart, and these may occur intermittently for years before the acute attack appears. These prodromal symptoms frequently occur with conditions that upset the vasomotor balance, such as worry, sleeplessness, fatigue, and constipation, and occur commonly in the morning. Vision becomes temporarily misty for a short time, rings appear around lights, and localized headache is frequent. The eye reveals a pericorneal flush, a steamy cornea, usually a dilated pupil, and raised tension. These cases usually pass into a chronic congestive glaucoma or into an acute crisis or real attack. In this we find the patient in intense unilateral pain, radiating through the head and most intense along the trigeminal nerve. There is nausea and vomiting. Lids are edematous. The bulbar conjunctiva and scleral vessels are much congested. The cornea is steamy, the anterior chamber shallow, the pupil partly and irregularly dilated and fixed to light. Vision is greatly reduced, due largely to clouding of the media and disturbance of the retinal circulation, and blindness may result in a very short time. The dilated and irregular pupil may be the one sign to distinguish the condition from secondary glaucoma due to iritis, in which case the pupil is contracted.

Acute iritic symptoms are usually gradual, acute glaucoma sudden. The differentiation, however, is sometimes difficult even for competent ophthalmologists, requiring history taking, experience, etc. to make a proper diagnosis. The eye grounds can not usually be seen, but if seen the optic disc is hyperemic. The tension of the eyeball is much increased. Sometimes acute glaucoma is mistaken for acute conjunctivitis. The history and symptoms are entirely different. In conjunctivitis the pupil is not dilated and fixed and there is usually pus, and there is no increase in tension. The physician can compare the tension of the two eyes with his fingers, and it is not difficult to note the difference in these acute cases. When taking the intraocular tension by palpation have the patient look down, then alternate pressure is made on the globe through the lids by the two index fingers, and so a relative idea of the resistance of the eyeball can be obtained. In acute purulent ethmoiditis with rupture into the orbit, which is rare, pain may be severe, but if the nose is examined the nostril will be seen to be blocked and pus will be found. Do not mistake acute glaucoma for a bilious attack or stomach or gall-bladder trouble.

CHRONIC INFLAMMATORY GLAUCOMA

Chronic inflammatory or chronic incompensated glaucoma occupies a position rather between the acute and chronic simple forms. The symptoms and inflammatory signs are much less than in the acute. The pupil is somewhat dilated and does not respond. Rises of tension may occur when the symptoms flare up. Cupping of the disc may occur quite early, contrary to the slowly progressive excavations of compensated. There may be an apparent physiologic cupping. If cupping is present it may be of the shallow saucer type, or the pressure may force the lamina cribrosa back and produce a typical pot-shaped glaucomatous cupping, extending to the temporal disc margin with vessels disappearing over the overhanging margins and reappearing on the floor of the excavation. This cupping description also applies in the chronic simple form. Visual field disturbances may not be present.

CHRONIC SIMPLE GLAUCOMA

Chronic simple or chronic noninflammatory glaucoma is by far the most common form of the disease, and while it occurs more in hyperopes it occurs more in myopes than do the inflammatory cases and it is always sooner or later bilateral. There is no inflammation seen and no pain. Rapid loss of accommodation is significant; in other words, the near point recedes. Field changes indicate actual presence of the disease and before the patient knows it there is contraction of the field, sometimes to almost a pinpoint, with good central vision still present. At first the pupil is normal both in size and reaction. One should go into the history of the case. When accommodation becomes uncertain, and

especially when there is rapid loss; when it is difficult to see after dark; when there are occasional light streaks, or when rings are seen around artificial lights; when there is temporary clouding; when there are headaches during the morning hours, or after an hour in a darkened room—all these suggest a transitory increase in intraocular pressure. Increase in pressure may also be caused by emotions, by excesses, by physical disturbances. An increase in tension to 38 mm. Hg. was found after a mountain climb (Gradle). Focal infection may produce a rise.

The preglaucomatous states are important to recognize, and require general care and medical treatment. When these preglaucomatous states are present and the tension lies on the borderline and the diagnosis is in doubt, provocative tests may have to be employed. The procedure might be as follows: Take tension, of course with a tonometer, then keep patient in a dark room and take tension again with as little light as possible—10 to 40 mm. Hg. rise. The patient may be given one or two cups of strong coffee—15 to 25 mm. Hg. rise in from 20 to 40 minutes. A good plan is to have a nurse, trained in tension taking, take tension every 2 hours at the patient's home. Tension is highest in the morning, but not much in normal eyes, which do not normally vary much during the day. If these tests are negative, then instill a drop or two of euphthalmine, and if tension is not increased in 3 or 4 hours it may be assumed there is no glaucoma. The writer has used 2% homatropine solution for this purpose, followed with eserine, and has never had any unfortunate effect from it. Tension, unless it is carried out somewhat as described above, is not the most important thing in arriving at a diagnosis of glaucoma. Green of San Francisco has obtained good results in scleral trephining in cases of keratoconus, and has come to the conclusion that the tension of that particular eye has been too much for it, although measured by the tonometer it may or may not be high. He believes that a given eye with a high refractive error, combined with marked muscular insufficiency and with poor ocular drainage, will, under pressure on the globe by the recti muscles and the lids, give way through the cornea. In other words, he considers keratoconus a form of glaucoma in which the pressure is exerted against the cornea rather than the optic disc.

The normal tension of an eye is that pressure which the tissues of the eye are able to withstand without impairment of function or damage to the tissues. The tension tolerance of the eye has been exceeded if repeated measurements show increasing abnormalities of the central field or contractions of the peripheral field or decreasing central visual acuity. Progressive loss of function due to hypertension is manifested first by abnormalities in the central visual field, by contraction of the peripheral visual field, and finally by decrease in central visual acuity. Of course, look for cupping, but don't wait for cupping before making a diagnosis. Optic atrophy and absolute glaucoma come later.

A careful map of the field of vision in glaucoma is very important. A nasal contraction, a partial or complete loss of the nasal field or of the upper or lower quadrant of the nasal side, very frequently is one of the earliest changes. This is explained by the fact that the blood supply from the central retinal artery is carried by a longer and more cir-

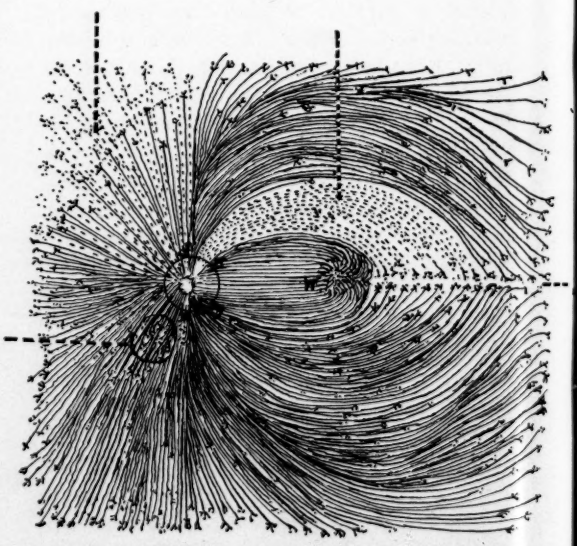

FIG. 1. Schematic representation of the topography of the nerve fibers of the retina. From Troncoso's "Internal Diseases of the Eye and Atlas of Ophthamology." By permission of the publishers, F. A. Davis Company, Philadelphia.

cuitous route to the temporal than to the nasal side, and the nerve fibers are decidedly arched as is shown in Fig. 1, and in B. T. Lang's specimen in Fig. 2 which beautifully shows the course through the distribution of persistent medullary nerve fibers. One, however, should not be satisfied with the examination of the peripheral field with a perimeter. A careful examination of the central area and blind spot should be made on a tangent screen, or the blind spot may be outlined on a campimeter. Seidel's scotoma consists of a wing-shaped extension of the blind spot upward or downward or both, and is one of the earlier signs. This progresses into the arcuate or Bjerrum scotoma and extends and ends at the horizontal raphe, and results in a Roenne step which is produced when either the upper or lower arching nerve fibers are most damaged, the inequality producing a step. This step is usually late. A Bjerrum may begin as a scotoma above or below the point of fixation. Should a double Seidel's and Bjerrum's scotoma extend along the arcuate fibres until its ends unite it is called an annular scotoma. Advanced cases show a field with a small central area and sometimes a large temporal patch. Central areas may be contracted to 10° or less and vision still be good. This has been called gun-barrel or tubular vision. There are other scotomas but those mentioned are the ones usually found in glaucoma.

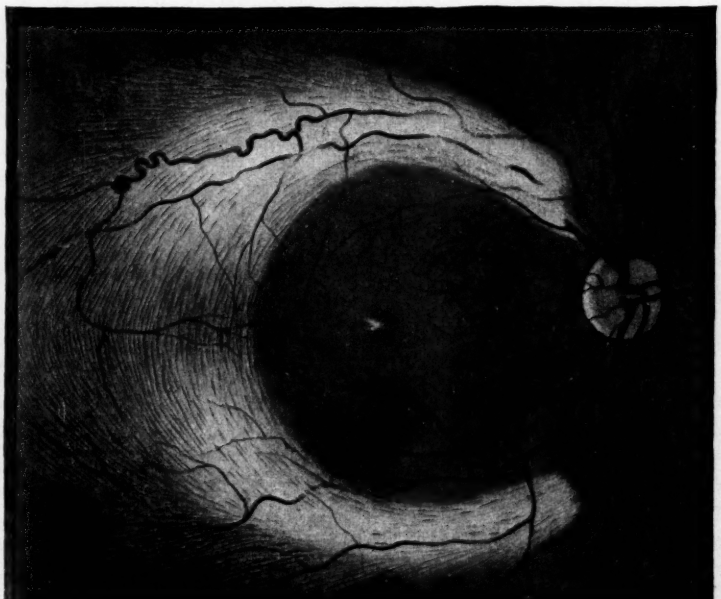

FIG. 2. An unusual case of medullated nerve fibers, which also illustrates the normal distribution of the retinal nerve fibers. From Peter's "Principles and Practice of Perimetry." By permission of the publishers, Lea and Febiger, Philadelphia.

It might be asked why does the macula escape? There are a great many nerve fibers supplying the macular area. The papillomacular fibers in the nerve head comprise about one-fifth of the entire nerve and are distributed to a very small area, and so a sufficient number may remain intact to preserve useful central vision until the last. In chronic inflammatory glaucoma, however, central vision becomes involved fairly early. Waite, Darby and Kirk have reported that the rate of dark adaptation is invariably retarded in the glaucomatous eye, and early in the disease. It can be tested by the examiner comparing his rate of adaptation with the patient's on the minimal readings of a foot-candle meter after both have set five minutes in the dark room.

Report of a case to illustrate the importance of an early diagnosis, and to illustrate the procedure in diagnosis:

Mrs. C., age 62 years, came to me July 24, 1939, for refraction. I had never refracted or examined her eyes before. Vision without glasses was 6/30 for right eye and 6/50 for the left. With her glasses, which she had had five years, her vision was 6/15 for right eye and 6/10 for left. She had compound hyperopic astigmatism. There was a need for a change of lenses, which correction brought vision up to 6/6 for each eye. The anterior chambers were observed to be rather shallow and by palpation the tension was increased. Pupils were round, equal, about 4mm., and responded to light and convergence. There was some bending of vessels over the margins of the discs. Tension was 32 mm. Hg. for each eye. After the patient spent ¾ hour

in a darkened room with her eyes closed, however, the tension rose to 42 mm. Hg. in each eye. This is far more than the increase in a normal eye would have been. Visual fields

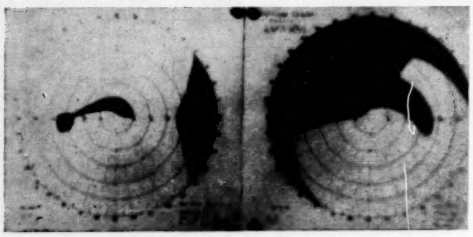

FIG. 3. Fields of patient taken July, 1939.

with a perimeter showed a loss of the upper nasal quadrant of the right eye and a Bjerrum scotoma of the left. A little later the tangent screen showed the picture seen in Fig. 3. The scotoma in the right eye had extended above the arcuate fibers to the periphery (Roenne's nasal step). The left eye showed a Bjerrum scotoma. A corneosceleral trephining was done on the right eye on November 7, 1939. A weak solution of pilocarpin is now being used. Tension runs between 28 and 32 for the two eyes, vision remains normal, and the fields have not changed.

CONCLUSIONS

Acute inflammatory glaucoma should be diagnosed and treated early because blindness may result in a few days. Do not mistake the condition for iritis and instill atropine. In iritis the pupil is contracted, and posterior synechiae may be present. Do not mistake this glaucoma for conjunctivitis. The early recognition of chronic glaucoma has been stressed because this form comprises about 90% of the cases. The later stage, where one can diagnose by questioning the patient or by feeling the eye and not even looking at it, has been omitted; i. e., the stage when the pupil is dilated and fixed, when the eye is hard, the fields contracted, and vision reduced or gone. Attention also should be called to the mistake made particularly by optometrists of calling glaucoma cataract. They are fooled because there is no inflammation seen, no pain, and vision is gradually reduced or lost entirely, and by the time that happens in one eye the other eye has usually advanced to such a stage that that is hopeless. Refractionists should test reactions of the pupils, should note the depth of the anterior chambers, the appearance of the eye grounds, and palpate for tension. Some opthalmologists take the tension with the tonometer of all patients past 40 years of age who come for refraction.

It should be repeated that progressive loss of function due to hypertension is manifested first by abnormalities in the central visual field, next by contractions of the peripheral visual field, and finally by decrease in central visual acuity; and that the tension tolerance of the eye has been exceeded if repeated measurements show increasing abnormalities of the central field or contractions of the field or decreasing central visual acuity. Degeneration of the nerve fibers may be arrested, but atrophy that has taken place cannot be regenerated, and when once optic atrophy starts it usually continues. Untreated glaucoma invariably leads to blindness.

There is another side that is of importance. If there are 30,000 people in the United States blind from glaucoma and it costs $1.00 a day to maintain them, the annual cost would be more than $10,000,000. If glaucoma is discovered in time and is given the proper treatment, it has been estimated that less than 8% become blind; so the 10 or 11 million dollars cost would be reduced to less than 1 million.

The family doctor is the first to see glaucoma, and if he does not recognize it the profession will have failed to that extent in its duties to the public, the country, and the profession. It is true that he does not have the armamentarium the ophthalmologist has, but he does not need it for his purpose in spotting early cases. He still has his tongue and his eyes and his finger tips which, as Gradle says, were all our forefathers in ophthalmology had.

DISCUSSION

DR. W. E. BIRD (Wilmington): I want to make one comment. In the Journal of the A. M. A. for September 29th there is described a very interesting case of glaucoma treated by a presumably competent oculist. Because he used his fingers to determine the tension he failed to diagnose the case as glaucoma. The patient went to another doctor who made a diagnosis with the use of the tonometer. The patient was given a diagnosis

of glaucoma. He later brought suit against the first doctor. The court decided, because the first doctor had used his fingers instead of the tonometer, that he was negligent in his practice and the patient was awarded a good round sum as damages.

DR. E. R. MAYERBERG (Wilmington): I would like to discuss Dr. LaMotte's paper. It may not be terribly interesting to most of you because of lack of interest in eye cases generally, however, his paper was magnificent.

He has done a lot of work on it and has presented it in excellent detail. Dr. LaMotte did not advocate taking tension only by the ophthalmologist. If the general practitioner suspected it he should, in all cases, particularly with eye tension, take the tension with a particular eye instrument. If we have a patient we suspect of being glaucomatous we take the tension.

Acute glaucoma is generally easy to diagnose. The patient notices the red eye and the terribly penetrating pain, not especially in the eye, but in that side of the head. As glaucoma is frightfully inoperative in the early stages every effort should be made to reduce the tension first. For two or three days use mydriatics every half hour or until the tension begins to decrease. Four or five days after the acute symptoms have subsided operative procedure may be instituted. Great care should be taken in operating: if the corneal section is made too rapidly the fluid gushes out and the lens is very apt to fall out too. Slow puncture first, to release the tension, and tension is usually permanently relieved. You can't say whether tension will be permanently relieved or not. Sometimes the patient will have recurrent attacks of glaucoma. He will have inflammatory adhesions in chronic cases.

Dr. LaMotte brought out a very interesting fact. If you have patients complaining that they are gradually losing sight, are going to someone for refraction every two weeks, glasses changed every two or three months, and no pain, regard that patient as a case of potential glaucoma, although it may be only a cataract.

After the optometrist gets through selling two or three pairs of glasses he usually says go to an eye man. Then we find changes in the retina and in the optic nerve, but those cases are so far gone there is nothing that can be done about them. The optic nerve is destroyed.

If a patient has one glaucomatous eye the chances are, in a few years, the other one will be just about like that. Don't pay too much attention to the acute cases, but the chronic case, with difficulty in seeing at night, difficulty in reading, having to have glasses changed frequently, should be sent to a competent ophthalmologist.

MENINGOCOCCIC MENINGITIS*

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We are in the midst of a meningitis epidemic of major proportions. During the first few weeks of 1943 the reported cases in the United States exceeded any comparable period during the fifteen years such data have been available. During the first thirteen weeks of 1943, ending April 3d, 5,826 cases of meningitis were reported, this being six times the reported incidence for a similar thirteen-week period of 1942. These facts are mentioned not with an aim of producing alarm, but for the purpose of stressing the current importance of reevaluating our knowledge of the disease and its treatment.

Definition: Cerebro-spinal fever is a febrile infectious disease caused by the meningococcus, a gram negative diplococcus present in the nasopharynx in many well people, but also capable of invading the leptomeninges in sporadic cases and in epidemics. The disease of meningitis is characterized, commonly, by a comparatively sudden onset of headache, stiffness of the neck, vomiting, prostration, leukocytosis, general tenderness, photophobia, mental cloudiness, irregular respiration, herpes and an eruption, most frequently hemorrhagic. The first three symptoms of fever, headache and stiffness of the neck are usual. The others are less uniform in their occurrence.

Epidemiology: In the absence of epidemics of cerebro-spinal fever, its prevalence is difficult to determine. Meningococcic meningitis occurs in all large communities throughout the year as scattered sporadic

* Read before the Delaware Academy of Medicine, Wilmington, April 28, 1943.
† Surgeon, Marine Hospital, U. S. Public Health Service.

cases, but the characteristic seasonal prevalence is in late winter and spring. The epidemic and epidemic peak both occur in January, February, March, April or May.

The disease is encountered both in the Tropics and in the Arctic region. Records exist of outbreaks in all civilized countries, particularly those of the north temperate zone. Interestingly enough, there have been more epidemics and heavier sporadic incidences in the United States than in any other nation. This is particularly true if one rules out all of the military epidemics of Europe. It has long been noted that the assembling of troops in barracks or of any group of people in any closely confined quarters for extended periods predisposed to the development of cerebrospinal fever.

Though the sharpest outbreaks of cerebrospinal fever is not in large cities, the heaviest incidences both sporadic and epidemic is in the urban communities, rather than in the rural. It may be said, therefore, that it is somewhat more characteristically a disease of the city where, perhaps, closer personal contact and poorer housing and living conditions may be an important factor. There is a greater prevalence of meningitis among males than among females, probably due to the greater frequency of massing large groups of men together. About half of the cases may be expected under the age of five years, but it is also quite prevalent during the early adulthood.

Predisposing Causes: The influence of age and of the assembling of large bodies of men in the barracks has been noted. In regard to the latter, two considerations may be of importance, intimate contacts as attested in the transfer of infection and the imposition of unaccustomed tasks causing a reduction of individual resistance. It is true that merely the assembling of rather large bodies of young men does not necessarily result in the outbreaks of meningitis as is observed at the beginning of the school years or colleges, it is seldom that one notes an outbreak of meningitis. However, these young men are not living in crowded sleeping compartments nor are they subject to any great fatigue. The question of over-crowding of living quarters is probably of paramount importance. This statement is born out by the findings of the Baltimore Health Department. In a city survey of overcrowding, 4% of the population were said to be living under such conditions. In a study of the homes in which meningitis cases occurred, 1300 were classed as "overcrowded households." Cold and damp weather is thought to predispose to epidemics. However, it is certain that many outbreaks have not been correlated with particularly low temperatures or high humidity. There is no noteworthy relationship to explain other non-specific respiratory infection and the onset or prevalence of meningitis.

Manner of Spread: Cases of cerebrospinal fever usually occur, but sporadically and in epidemics, without demonstrable direct contact with other cases. Marked maps show a scattered distribution throughout the localities affected. Doctors, nurses or other patients do not ordinarily contract the disease from cases in the hospital. It is rare for more than one case to occur in a family, even though contact has been most intimate between the sick individual and other members of the family prior to the making of the diagnosis. The explanation for this peculiar distribution of an infectious disease is not easily made. Up until a few years ago it was felt that as long as the carrier rate in the general population did not exceed 20% there was little likelihood of an epidemic. Such views are not now held to be valid. Various studies have shown the carrier rate to range between 1% and 50% of the studied population group. The results are practically uniform, however, that persons who have been in contact with cases of meningitis are more likely to be carriers than those who have not, but many people who harbor the organism have had no known contact with the disease. Dudlay and Brennan noted that at the Royal Naval Hospital in Catham, England, a carrier rate of 50% for agglutinable meningococci persisted for over a year without the appearance of a single case of meningitis; whereas at the Royal Naval Hospital at Portsmouth, during the same period, the carrier rate among contacts of sick patients with meningitis was only 5%. At the present time the significance of carriers and carrier rate is difficult to evaluate. Because of the high carrier to case ratio it

has been suggested that "the carrier constitutes the mass of the epidemic and the cases are an accidental minority."

Bacteriology: The specific etiologic agent of meningococcic meningitis is, of course, the meningococcus. It is an organism easily stained by the ordinary basic aniline dye, but loses its stain when decolorized by Gram's method and takes a counter stain instead. It may be found during the course of the disease in the blood stream, the nasopharynx, in the cerebrospinal fluid and in the petechial and ecchymotic rash on the skin. Nothing need be said concerning the morphological or cultural characteristics of the organisms in this paper.

The meningococcic organisms have been classified and grouped by various investigators, but with our present method of treatment, aside from academic interest, there is probably little of practical value gained by typing in a given case. Most epidemics are due to Group I meningococci. Some outbreaks have been due to Group II. Therefore, from the standpoint of epidemologic study it might still be important to type contacts during an epidemic, but seldom as an aid to therapy. There is no reason to doubt that the meningococcus reaches the body and leaves the body by way of the nasopharynx where it is commonly found.

Symptomatology: Clinical history — Our former ideas of a more or less definite period of incubation for each infectious disease have been modified. In general the more highly contagious disease, the more definite the period of incubation. Because of the great variability of the apparent virulence of the meningococcic organism, it is difficult to determine any period during which an individual is most likely to develop clinical symptoms. The belief is held by some writers that every case of cerebrospinal fever is first a carrier and that by breaking down perhaps temporarily of the defense mechanism, the organism is able to invade the body and cause the disease. In instances where carriers have been isolated, clinical cases have occurred varying periods from their time of segregation ranging from one to forty days. Of course there is no way of knowing just how long these people had harbored the organisms in their nasopharynx. In a study carried out in the British Navy, 485 carriers were kept under observation during an outbreak of meningitis, but not one carrier developed it.

On the rare occasions where more than one case occurs in the same family or among mess mates, among the troops, the period of onset may be separated by hours or months. The possibility of both cases being infected from a common source must always be borne in mind. More definite are the cases which develop in a previously non-infected district, after a visit to or from an infected focus. Such evidence places the usual incubation period in one to five days.

The clinical picture presented by cases of patients with cerebrospinal meningitis is varied. Clinical manifestations are those of a generalized infection and of meningeal irritation. The onset in a typical case is sudden with malaise, fever, nausea and vomiting, chills, headaches and a stiff neck, with the other evidences of increased intracranial pressure. Nasopharyngitis may or may not proceed the illness, but if present, is usually due to some organism or virus other than the meningococcus. Skin manifestations, especially petechial hemorrhages and other forms of rash are found most often during epidemics and may occur in from 10% to 70% of cases. Patients with a fulminating form of this disease may present the picture of a collapse associated with Waterhouse-Friderichsen syndrome. In a small percentage of cases the presenting symptom may simulate acute polyarthritis or as a recurrent gastrointestinal disorder.

Mode of Onset: The onset is as a rule such that the patient has had enough fever and prostration to be kept in bed within 24 hours of the first clear symptom. Ambulatory cases with true meningeal headache are unusual. A normal temperature may be retained for several days and may delay the diagnosis. One must not overlook the fact that in certain very virulent cases the individual may retire feeling well, but die of the disease during the night, the first intimation of the cause of death being found on postmortem.

A chill with rigor or a convulsion in an infant frequently ushers in the disease. The chill may be replaced by a chilly feeling and

malaise or this period of invasion may be so masked or so soon succeeded by definite meningeal symptoms or unconsciousness that it escapes notice. The convulsions are not usually prolonged beyond the first day and they are very rare in adults. Vomiting occurs in more than half the cases at or soon after the onset. Through this symptom, together with a not uncommon abdominal pain in children, often directs attention to the gastrointestinal tract. The vomiting usually is not proceeded by nausea and frequently bears no relation to meals. The vomiting is a symptom of the initial stage and is not usual thereafter in contrast to other cerebral diseases.

Objective Symptoms: Usually the first definite meningeal symptom in the adult is a headache. This may be referred to any or all parts of the cranium. Frontal headaches frequently give way to occipital as the disease progresses. It cannot at first be differentiated from an ordinary headache accompanying any general infection or toxemia; the headache of cerebrospinal fever typically becomes an intense one with most persistent symptoms. The headache frequently becomes one of the most prominent symptoms unless supplanted by delirium or mental dullness or unless relieved by treatment. It is not stopped by ordinary methods. If the patient knows by experience the improvement following lumbar puncture, he may beg for the latter. The fact that this characteristic symptom is definitely absent in some cases is to be remembered.

The mentality at onset and in the early stages may be normal with allowance for the prostration, pain and mental apprehension. Commonly the patient is either hypersensitive and irritable or dull and unresponsive, though in the epidemic cases there may be delirium, stupor or collapse when first seen. During the height of the disease a case of average severity may be expected to be deeply stuporous with some delirium, but without complete coma. Vertigo or double vision is occasionally found.

Physical Findings: The most important sign of meningitis is stiffness of the neck or resistance to flexion. In eliciting this sign the examiner should proceed with all gentleness, especially in the cases of children. It is well to secure the history and make other less important and less painful examinations first in order to gain the confidence of the patient. Rotation of the head from side to side should be attempted, though lateral rigidity is not so significant as antero-posterior. To determine the latter, with the patient lying on his back, the hand of the examiner should be inserted between the occiput and the pillow, palm up and gently raised. Unless the condition is marked, one examination is not sufficient, but another attempt to flex the head should be made at the conclusion of other tests. Unlike a rigidity of less serious import, a neck rigidity of meningitis does not loosen up on manipulation. The stiffness of the neck may be so marked that some retraction or opisthotonos is visible.

Of almost equal importance with the information obtained by attempting to bend the neck is the phenomena known as Kernig sign. This is a resistance of passive simultaneous flexion of the hip on extension of the knee. Any limitation of extension to an angle of 135 degrees or less at the knee may be considered a positive Kernig. It must be remembered that in elderly people the positive Kernig sign is normal and not indicative of disease.

It is well to do general neurological reflex studies although they are not of great help in the diagnosis of meningitis. Deep reflexes are usually exaggerated at the onset of the meningeal involvement. The superficial reflexes are dulled, but often changes occur in the opposite direction.

A more important finding in the patients who have a severe degree of illness, who are still conscious, is the generalized hyperaesthesia in the skin and the deeper structures which is present in many cases. The secondary effect in this generalized tenderness is apprehension, irritability. The patient shows a disclination to be handled or to be moved. Ophthalmoscopic examinations usually gives little information. Pupils are usually dilated and react sluggishly. They may be unequal or they may be contracted. Deafness is not infrequent in the disease and is often central in origin.

Temperature in cerebrospinal fever at the onset is typically high, but there is nothing constant or diagnostic in the chart, although

irregularities are very common; temperature is a poor guide in condition or prognosis. Pulse is often rather slow in proportion to the temperature and shows varations independent of the temperature. This is, of course, the result of changes in the intracranial pressure.

The most frequent skin manifestations in cerebrospinal fever, probably is herpes, occuring around the mouth and nose. It usually does not appear before the third day. It may occasionally be widespread. Herpes has no prognostic significance. Typical eruption of the disease is hemorrhagic, the spots varying in size from those of flea bites to enormous splotches in different cases, showing much variation in the individual case. The eruption is usually in sporadic cases. In many epidemics it is observed in less than 50% of the cases. The cases showing eruption are as a rule more severe than those without an eruption. However, the mortality difference is not great.

Diagnosis: The diagnosis of meningococcus meningitis depends upon the findings of some or all of the above mentioned signs or symptoms and additionally one usually finds an elevated white blood count which may range from normal up to 25 or 30,000 with a very high polymorphonuclear-neutrophile count. Some cases may show 95% to 97% polymorphonuclears to be present. However, one must not withhold a diagnosis simply because the count is not markedly elevated. We have seen cases of meningococcic meningitis with counts ranging from between 8 to 10,000.

Lumbar puncture is probably the most important single diagnostic procedure done to determine accurately the diagnosis of meningococcis meningitis. It may be performed for two definite reasons. First and most important is the examination of the spinal fluid to determine the organism present, the cell count and the various chemical properties of the spinal fluid. The other reason, less important but often helpful, is following the course of the disease from a menometric determination of the spinal fluid. The normal range in the adult is generally from 100 to 150 mm. of water, depending to a certain extent on the degree of relaxation of the patient during the operation. The initial pressure reading of persons when first seen with meningitis may go above 500 mm. of water. In these cases with high pressure care should be exercised in the removal of fluid. It should be removed slowly and not in amounts that would lower the pressure below one-half of the original reading. The pressure is not always markedly increased. In some cases of coma or those in a wild maniacal state one may have a spinal fluid pressure of only 250 mm. of water.

The cell count very early in the course of the disease may be low. It may be possible early in the disease to find by cultural methods the presence of the meningococcic organism in the spinal fluid and still have no increase in the number of white cells present. In these instances the pressure is most always elevated. However, as a rule at the time that the lumbar puncture is done, one finds an opalescent or cloudy fluid with the cell count ranging from a few hundred up to two or more thousand cells. On spinning the spinal fluid in a centrifuge and making a direct smear from the sediment, one can in the majority of instances find gram negative intracellular diplococci present. He is then in a position to make an almost certain diagnosis of meningococcic meningitis. However, aside from the treatment, it is always well in the laboratory to culture a sample of the spinal fluid and to run the organism recovered through sugar to determine exactly the nature of the gram negative organism. We know that meningococcus, gonococcus and catarrhalis all have a similar morphological appearance. This, however, is not waited for by the clinician. As soon as the presumptive diagnosis of meningococcic meningitis is made he should institute treatment.

A word should be said about the advisability of doing lumbar puncture in the presence of a severe rash without stiffness of the neck or any other manifestations of meningeal irritation. There have been two schools of thought on the matter and probably the question is still an open one. There is a belief based on careful clinical studies that when spinal punctures are done on bacteremic patients who do not have a meningitis a meningeal infection will follow. On the other hand, Frey recently reviewed 207 cases of

pneumococcic meningitis in order to determine whether lumbar puncture was a factor in the pathogenesis of its development and concluded that "The incidence of meningitis is no greater among patients with bacteremia who were subjected to lumbar punctures with the yield of normal cerebral spinal fluid than among those for whom no diagnostic tap was performed." The same conclusion can probably be applied to meningococcic meningitis although proof is lacking. Dingle and Finland believe that blood is cerebrospinal fluid for bacteriological examination should be obtained immediately from every patient in whom meningitis is suspected. They base their opinion on the following consideration:

1. "There is no clear proof that lumbar puncture results in meningitis, the patient in whom meningeal infection would not otherwise have developed."
2. "With proper precautions the procedure does not in itself endanger the lives or subsequent recovery of the patients."
3. "Clinical diagnosis alone is not sufficient fool-proof since some patients may have definite, even extensive, meningeal infection without the pathognomonic signs of such involvement."
4. "Early diagnosis followed by prompt and adequate therapy increased the patient's chances for recovery."
5. "Any risk of inducing meningitis, if it exists at all would be minimized by sulfonamide which would undoubtedly be instituted in almost every case in which such an infection occurred."

It is felt, therefore, by most of the workers in the field at the present time that the dangers of waiting to make a diagnosis of meningitis outweighs the dangers from doing a lumbar puncture with the theoretical possibility of introducing the organism into the spinal fluid by virtue of the diagnostic procedure.

Therapy: The treatment of meningococcic meningitis falls under three headings:

1. Chemotherapy
2. Serotherapy
3. Symptomatic treatment and treatment of complications.

Chemotherapy: Soon after the recognition of the effectiveness of the sulfanilamide against the hemolytic streptococcus, Saddle, Gray and Stevenson reported good results from its use in experimental meningococcal infection in mice. Since that time in 1936 sulfanilamide and several of its derivatives have been so successfully employed in treatment of cerebrospinal fever that they have largely replaced serotherapy. While sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine have all been found therapeutically effective when used individually or in combination with serum, it is at present generally agreed that sulfadiazine because of its low toxicity, ease of administration and uniform effectiveness is the drug of choice administration. Sulfadiazine may be administered orally, intravenously or subcutaneously. The oral route is the method of choice, but in meningitis, with or without coma, it is important that an effective therapeutic blood level be reached as quickly as possible. In such instances sodium sulfadiazine is especially useful. It can be administered intravenously as a 5% solution in distilled water or in normal saline. It is excreted by the kidneys more slowly than is either sodium sulfapyridine or sodium sulfathiazole. Because of this fact it is possible by repeated injections at 8-hour intervals to maintain a satisfactory blood level. Oral administration of sulfadiazine should be started as soon as it is possible for the patient to swallow the tablet. In instances of coma when the sodium sulfapyridine is not available, the drug may be given by injecting a suspension of the crushed tablet through a nasal tube. This is a very useful procedure at times and may be lifesaving. Sodium sulfadiazine may also be given subcutaneously in a 0.5 to 2% solution. The sodium salts are quite alkaline and are extremely irritating. If they are injected outside the vein, the solution having a concentration greater than 2%, they would cause local tissue damage. They must never be used intrathecally as their alkalinity will cause irreparable damage to the nerve tissues.

Dose: The initial dose of sulfadiazine for an average 150-pound man should not be less than 4 gms., preferably 5 gm. This should be followed every four hours by one gm. or every six hours by 1½ gms. If one wishes to

continue intravenous administration for the first 24 hours after the initial injection of 5 gms. of sodium sulfadiazine, the patient should be given 2 gms. every 8 hours. At the end of 12-18 hours, when possible, a blood level determination should be done. It may be found necessary to increase the size of the oral dose or to add an extra gram or two of the drug intravenously to bring the level to a satisfactory therapeutic point. A similar dosage scheme is followed for subcutaneous administration.

Blood Level: There does not appear to be a universally effective blood level because of individual variations in response to the drug. Some patients appear to do well and a cure is effected when the blood concentration does not exceed three or four mgs. per 100 cc., while others appear to require 8-10 mgms. per 100 cc. Sutides have revealed that on an average, a concentration of the sulfadiazine in the spinal fluid is only 50 or 60% of that in the blood. It is, therefore, good practice to keep the blood level in a case of cerebrospinal fever about 12-15 mgs. per 100 cc. It should be mentioned that when first introduced into clinical use, sulfathiazole was not recommended for treatment of meningitis because it was found in relatively low concentration in the spinal fluid even in the presence of a blood level of 8 or 10 mgms. per 100 cc. In spite of the early predictions its use has been followed by excellent clinical responses and cures. It is possible that the actual concentration of the drug in spinal fluid is not of great importance, but in order that one may more accurately evaluate his results, both blood and spinal fluid determination should be made at least every other day during the early days of treatment.

How long should the drug be continued is always a question that confronts the physician. It is frequently difficult to answer this question and say when a patient has recovered from his disease or to know what clinical data to use as a guide in discontinuation of therapy. Within 24 hours after chemotherapy has been instituted some patients may be afebrile, have a normal pulse rate, normal blood pressure and to all appearances he has recovered. However, if his spinal fluid is examined at that time, it may be loaded with pus cells. A safe rule to follow is to continue full doses of sulfadiazine until spinal fluid is clear, then reduce the dosage to one-half for another four or five days. If some such scheme is not followed, there will be many serious relapses or recurrences. Patients who have recurrences should be treated as a new case, although a warning should be sounded relative to the follow-up of blood counts; all patients who have been on chemotherapy for one week or longer should have at least two blood counts, five to seven days apart after discontinuing the drug. This may reveal a developing blood dyscrasia that would not otherwise be discovered at early stages.

Serotherapy: The treatment of cerebrospinal fever with serum has largely been supplanted by chemotherapy. In Circular Letter No. 17, issued February 23, 1942, by the Office of the Surgeon General of the United States Army, anti-meningococcic serum was not recommended unless deemed necessary in the individual case. While it is true that chemotherapy alone is usually adequate, there are instances in which serotherapy is recommended. It would seem appropriate, therefore, to reserve the use of anti-serum as a supplement to chemotherapy for the very severe or the apparently fulminating cases. It is particularly recommended for patients in coma, in whom bacteremia is suspected. In other cases anti-serum should be employed when there is no definite objective evidence of improvement after 24 to 48 hours of adequate chemotherapy. It is also indicated in those cases manifesting drug intolerance. The relative value of anti-toxin and anti-bacterial serum is still undecided. Hoyne advocates the use of anti-toxin although experimentally it appears to show no properties other than those due to anti-bacterial immune bodies that it contains, but serotherapy if employed alone or as a supplement to chemotherapy should be administered in adequate doses. To the average 150-pound adult with meningitis an initial dose of 150 to 300 cc. of anti-meningococcic serum should be given and repeated within 24 hours if no improvement is noted. Hoyne and others are advising against intraspinous therapy and are recommending only the intravenous route. If anti-toxin is to be

used a dose of 60,000 (180 cc.) units to 100,000 (300 cc.) should be given and repeated in 12 hours. — Hoyne has given as much as 1200 cc. within 4 days and reports excellent recoveries without complications.

In either instance the anti-toxin or serum should be diluted with 10% dextrose in saline solution of at least equal volume, but preferably double on more than the volume. It is important to have the dextrose in the saline solution to avoid the precipitation of the serum. It is also advisable to add to this mixture .05 to 1 cc. of epinephrine and administer at body temperature by the drip method. The flow should be started slowly, but after a few minutes it may be increased to 60 drops per minute.

Symptomatic Treatment: Symptomatic therapy in the treatment of meningitis is of great importance, but unfortunately frequently overlooked. It is important that the fluid balance should be kept at a good level. In febrile patients who are in a semicomatose condition, too often they are allowed to become dehydrated. They should be given at least 3,000 cc. of fluid a day and probably not more than 3,500 cc., and it should be admonistered intravenously or subcutaneusly if the patient cannot take this amount by mouth. The food intake is not of great importance because with the present therapeutic measures most patients either recover or succumb to the disease before the nutritional state would make any great difference. It is well, therefore, to give the patient sweet fruit juices or 5% to 10% intravenous glucose, but other than this one need not worry in the majority of instances. It goes without saying that a sponge bath in cases of high fever, an ice cap to the head for relief, sedations for relief of restlessness, lumbar puncture to relieve the spinal fluid pressure in those cases who build up high presure with an accompanying restlessness and delirium are all factors of importance. In the comatose state, one must be ever on the alert to se that the patient's bladder is emptied regularly. An incontinent patient may have a tremendously enlarged bladder extending almost up to the umbilicus and his incontinence merely representing an overflow. This is an important point in watching comatose cases.

Sedation in the meningitis patient is frequently one of the most difficult problems to answer that confronts the physician. In a delirious, maniacal patient one may give tremendous doses of the barbituric acid preparations intravenously without causing much sleep or relaxation. Morphine is not recommended as being a particularly good drug to quiet these cases. Some of our neuro-surgeons feel that in the presence of brain injury morphine should not be given at all. Pentothal or sodium nembutal intravenously has been recommended by some and rectal anaesthesias of avertin have been used to bring about relaxation. The writer has had patients who have been delirious in which it was practically impossible to quiet them with any drug used. It should be said that intravenous pentothal was not tried.

Complications and Sequelae: The incidence of complications and sequelae have been greatly reduced since the advent of chemotherapy. The shift in the mode of administration of serum from the intraspinous to the intravenous route has probably reduced the incidence of central nervous system complications also. One notes that prior to chemotherapy the following complications were reported as occurring: "There may be cranial nerve involvement, particularly deafness and blindness, changes in mentality, transient paralysis, convulsions, encephalomyelitis or acute encephalitis, panophthalmitis and other types of ocular infection, arthritis, purpura, pericarditis, endocarditis, myocarditis with cardiac enlargement, pleurisy, orchitis and epididymitis, transient albuminuria and hematuria, adrenal hemorrhage and diabetic coma." To be sure, probably any of these complications may still occur, but perhaps less frequently. However, arthritis and herpes occur in an appreciable number of early and adequately treated cases. One must also be on the alert for complications resulting from chemotherapy and when serum is used for various manifestations for serum reaction. The complications resulting from drug therapy may be nausea and vomiting, cyanosis, drug rash, hematuria, hemolytic anemia, leukopenia and granulocytopenia, oliguria, flank pain or typical renal colic and other less important renal manifestations.

The incidence of these complications resulting from therapy is greatly reduced if sulfadiazine is used and if an adequate fluid intake maintained. An adult on the drug should void 2,000 cc. of urine daily as a minimum. Drug fever usually does not appear until the second week from the 9th to the 14th day, nor does leukopenia or agranulocytopenia usually occur until the second week. It is important, therefore, to give early and adequate drug therapy and stop as soon as possible, thus many of the later complications can be avoided.

Effectiveness of Newer Methods of Therapy: That our newer methods of treatment are an improvement over previous methods is quickly shown by the mortality statistics. The following figures were supplied by the Baltimore City Health Department:

Year	Cases	Deaths	Percent of cases that died
1917	122	58	46
1918	175	75	42
1931	42	16	37
1932	24	9	37
1935	182	63	29
1936	285	94	33
1942	201	31	15
1943 (1st 13 wks)	189	25	13

One can gather from these figures that our over-all mortality rate should not exceed 15 or 16% even in an epidemic period. During non-epidemic periods it should be less if early diagnoses are made and early, adequate therapy is instituted.

It is frequently difficult to determine whether a secondary rise in fever beginning at the end of the first week is due to the drug or to a recrudescence of the disease. Fever alone is no contraindication for continuing the drug. However, a search should be made for evidence of recurrence or complication of the infection. If none is found, it is well to discontinue the therapy if the patient's condition will warrant it; if not, change to another type of sulfonamide drug.

Complications resulting from serum are usually in the form of arthritis or urticaria. They may be treated symptomatically.

One of the most serious complications of meningitis is the peripheral vascular collapse resulting from adrenal hemorrhage (Waterhouse-Friderichsen syndrome). It is treated as any acute adrenal insufficiency by giving sodium chloride and adrenal cortical hormone. Normal saline should be given intravenously during the period of marked collapse, either intravenously or subcutaneously. One may add one cc. 1-1000 epinephrine subcutaneously at once and add 1 cc. to each litre of fluid given thereafter. Cortical adrenal extracts in 10 cc. amounts may be given every 2-4 hours or until improvement of the peripheral vascular state is noted, after which it may be given less frequently depending on the level of the blood pressure, the character of the pulse and the patient's general physical state. If this complication is not recognized and treated early and vigorously the patient may die within a matter of a few hours. On the other hand, there have been cases of recovery reported.

Meningococcemia: A word should be said concerning a meningococcemia, a condition which may occur as a disease entity without meningitis or other manifestations of central nervous system involvement. However, meningococci have been demonstrated by cultural methods in the blood of 25% to 80% of cases of meningitis, depending upon the cultural methods and the stage of illness at the time of taking the blood. In fulminating cases one may see the patient first because of generalized aches, arthritis, photophobia and generalized purpuric rash, without any manifestations of meningeal irritation. When lumbar punctures have been done early on these patients, spinal fluid is frequently found to be normal and there may or may not be any increase in pressure. It is particularly important to diagnose such cases correctly and institute therapy immediately before meningitis develops. It should be added, however, that in some cases no meningitis ever developed even without treatment.

Isolation of the Clinical Case: The period of isolation of cases of meningitis is often governed by the recommendation of the state or local health department. 14 days is the

usual prescribed period of isolation. The presence or absence of positive throat cultures is not a criteria to be used as a guide. With chemotherapy the patient may appear well, be afebrile, have clear spinal fluid, etc. by the end of seven or eight days. In spite of this it is still the practice to isolate the patient for the full fourteen days. The practice of segregating the actual case of meningitis is undoubtedly advisable, but the isolation of carriers is probably of limited value and should not be done.

The danger of contracting meningitis through contact with a case is probably much overstressed. The incidence of multiple cases in the same household in Baltimore out of 201 cases during 1942 was less than 3%. The question always arises—should one do anything about treating contacts. Most workers feel it is not necessary. However, sulfadiazine may be given to the contacts in doses of 4 grams a day for the adults. This does rid the carriers of their organisms. This is not believed at present to be a good practice for large groups of people. It is not being practiced in the Army where studies have shown 50% or more of the soldiers to be carriers.

Conclusion: This paper was written for the purpose of briefly reviewing the subject of meningococcic meningitis and to present some of the more generally held concepts relative to therapy. There are several argumentative points dealing with diagnosis and treatment which were only touched upon. It is felt, however, that the views presented are the ones generally subscribed to.

DEAR DOCTOR

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READ IT!

Advises Physicians to Inform Selves About New Social Security Bill

Physicians should inform themselves concerning the origin and objectives of the proposed Wagner-Murray-Dingell bill broadening the American social security program, *The Journal of the American Medical Association* for June 26 advises in an editorial discussing the measure. *The Journal* says:

"In its evolution the . . bill stems from the National Health Conference of 1937, the Wagner bill which followed that conference, and the report of the National Resources Planning Board. Essentially in its medical aspects it is a compulsory sickness insurance bill and an attempt to translate the proposals of the Social Security Board into a technic of action. Inquiry of reliable sources in Washington indicates the probability that the actual designers and authors of the bill included I. S. Falk, director of the Bureau of Research and Statistics of the Social Security Board of the Federal Security Administration, Mr. Wilbur J. Cohen, technical adviser to the Social Security Board, and Senator Wagner's secretary, Mr. Philip Levy. . . . Inquiry also reveals that as far as can be determined, representatives of the medical profession, either within or without the government, were not consulted in the development of the medical provisions. Evidence of this failure to consult the medical profession appears in the language of the proposed bill, since it speaks twice of a 'spell of sickness.' The word 'spell,' thus employed, does not appear in English dictionaries except as a colloquialism in Webster, and the term is seldom, if ever, used by any one educated in medicine. . . .

"Speaking bluntly. . . . the measure apparently attempts to avoid the numerous difficulties involved in developing a government controlled medical service by making the Surgeon General of the Public Health Service, whoever he might be, a virtual 'gauleiter' of American medicine. Indeed, it is doubtful if even Nazidom confers on its 'gauleiter' Conti the powers which this measure would confer on the Surgeon General of the U. S. Public Health Service. . . .

"In offering the bill its proponents emphasize that it provides for free choice of doctors; free choice of a doctor means of course, free choice of doctors willing to engage in this type of work. . . ."

Editorial

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MEDICAL STUDENTS ON ARMY PAY

The powers of the Federal Government have been the subject of controversy ever since the Constitution was ratified by the Original States. Hamilton led those who favored a strong central government, and Jefferson those who believed in local self-government with the Federal Government doing only what was beyond the powers of the States—foreign relations, postal system, etc. The balance of power between the Federal Government and the States has never been settled. It has been the cause of duels and of civil war. Through the years the central government has grown constantly stronger and that of the States proportionately weaker. Most of this gain has been brought about by constitutional amendments and legislative enactments. Each war accelerated the process. Rarely does the Federal Government relinquish any authority it has once gained, control over the railroads in World War No. 1 being a notable exception.

Even before World War No. 2, the government in Washington began to use a new technique in extending its authority. It began to use its tremendous monetary power. By offering grants or bonuses if the States would undertake certain measures in accordance with specifications drawn up in Washington, the Federal Government gained control of various functions of the States; notably roadbuilding, certain public health activities, and relief measures. Whether these measures are wise or not is beside the point. The fundamental principle involved is the important thing. If the trend towards centralization continues at the same rate, what will become of State Governments? Will they become merely historical vestiges like the urachus or the obliterated hypogastric arteries?

The Federal Government has recently extended its influence to the field of education. In this move they are using a two-edged sword —monetary power and the control of patronage. Except for girls and a few rejectees, it has absolute power over the youth of the land. Uncle Sam says to the able-bodied youth of the nation who shall go to school and what they shall study. The liberal arts colleges are hard put to justify their existence. In this total war such control is no doubt necessary. We hope that the love of learning will not be entirely suppressed. The U. S. S. R. seems to be able to take an interest in cultural affairs and at the same time do a pretty good job at waging war. It recently celebrated Sir Isaac Newton's 300th anniversary in seven different cities.

As to the future of medical education, Wingate Johnson in an editorial, "Quid Pro Quo," in the March issue of the *North Carolina Medical Journal*, paints a very gloomy picture. He points out that the recent provision for paying the medical student's tuition and maintenance, and allowing him fifty dollars a month pocket change is entirely unnecessary. There is not a medical school in

the country that has not for years turned away more applicants than they could take. There is nothing to indicate that this condition would not have continued if the prospective medical student had been given a deferred rating by the draft boards. There was no reason for paying him. There must have been an ulterior motive, and this, Wingate Johnson more than hints, is the control of medical education.—Editorial, *Va. Med. Mo.*, June, 1943.

WORKMEN'S COMPENSATION LAW

AN ACT TO AMEND CHAPTER 175 OF THE REVISED CODE OF DELAWARE, 1935, ENTITLED "DELAWARE WORKMEN'S COMPENSATION LAW," BY PERMITTING EMPLOYEES TO DESIGNATE THEIR OWN PHYSICIANS OR SURGEONS.

Be It Enacted by the Senate and House of Representatives of the State of Delaware in General Assembly met:

Section 1. That 6078, Section 8 of Chapter 175 of the Revised Code of Delaware 1935, as amended by Section 6 of Chapter 269 of Volume 43, Laws of Delaware (1941) be amended by adding thereto a new sub-section to be known as Sub-Section (f) as follows:

(f) Any employee may apply in writing to the Industrial Accident Board for permission to employ a physician or surgeon other than the physician or surgeon furnished by his or her employer. Such application shall state the name and address of the physician or surgeon whose services are desired by the employee. Upon the approval by the Industrial Accident Board of any such application, the employee by whom or on whose behalf the application was filed shall be entitled to receive from his or her employer the reasonable cost of the services of any physician or surgeon whose employment was approved, performed subsequent to such approval, within the limitations provided for in subsection (b) of this section.

Approved April 16, 1943.

How Color Blind Men Are Admitted to Navy and Army Air Forces

Discussing a technic which is claimed will educate men who are color blind so that they can pass the armed forces' tests for color blindness, *The Journal of the American Medical Association* for June 5 says that "Obviously, it is for the services themselves to determine whether or not the tests for color blindness now used are sufficient, in view of the possibilities of learning, to screen out enough persons who are color deficient or whether or not the young man who is willing to put in the necessary time in an educational process to learn enough about the tests to enable him to pass them would not qualify because of superior learning ability or intelligence to meet the needs of the armed forces.

"In *The Journal* for March 20 references was made to an alleged cure for color blindness said to have been devised by one J. H. Lepper, optometrist, of Mason City, Iowa. Subsequent to the publication of that statement, Lepper sent to *The Journal* a communication in which he insisted that he does control color blindness in from fifteen to twenty days so that boys who followed his methods passed government tests. He submitted complete instructions as to the technic that he uses, some special lenses which are used as a part of the procedure, and a list of men now in the Navy and in the Air Force who had previously been rejected but were accepted after having followed his technic.

"The material concerned was referred to appropriate agencies in Washington, which verified the information supplied by Lepper as to the men who had been rejected and later accepted following his attention. Investigators then visited certain optometrists in the eastern part of the United States who follow the Lepper technic and studied their results. These investigators believe that Lepper is conducting an educational rather than a therapeutic process. In his technic the subjects are instructed to get some one with normal color vision to assist them and to trace the shapes of the figures carefully even in those charts in which they can see the figures fairly well. They are instructed to continue working on harder and harder charts and to

keep daily records of the charts which they can read and those which they cannot read. Thus the person concerned builds a learning curve and with sufficient time masters the color manual thoroughly.

"It seems quite likely that those with disturbances of color vision do fix in their minds appearances which are associated with certain colors when viewing objects that have for them some familiarity. No doubt reaxamination of these individuals at a later date, using a different technic, would reveal that the persons concerned are still color deficient, although they did learn enough about the test ordinarily used to pass that test and thus enable them to enter military service. . .

"The optometrists who are now specializing in the Lepper technic might limit their promotion of the method to the statement that they teach men to pass the tests for color vision and avoid carefully the use of the word 'cure' in any relation to color blindness."

Tetanus Immunization of Military Personnel

All military personnel on induction are being immunized against tetanus either, as in the Army, by three injections of fluid toxoid, or as in the Navy and Marine Corps, by two injections of alum precipitated toxoid (*New Eng. J. Med.*, 227: 162, 1942). In addition a small or stimulating dose is injected prior to departure for a theater of operations and an emergency dose is given to those wounded or burned in battle or incurring other wounds likely to be contaminated with clostridium tetani. According to recent report (*Am. J. Pub. Health,* 33: 53, 1943) since June, 1941, when the present tetanus immunization program was adopted, there have been but four cases reported from the entire Army, and none of these were in immunized individuals. Although perhaps too early in the present war to draw any conclusions, it is of particular interest that no cases of tetanus have been reported from battle casualties.

For civilian use, especially in children, it is of decided advantage to accomplish simultaneous immunization against tetanus and diphtheria. Combined Diphtheria Toxoid-Tetanus Toxid, Alum Precipitated, Lilly, is designed for prophylaxis only, affords effective immunity against both diseases, and avoids risk of serum sensitization which may follow use of an antitoxin.

New Vitamin—Bc

Isolation and crystallization of a new antianemic vitamin—Bc—half a million times more potent than fresh liver, which may speed convalescence from many illnesses because of its blood regenerating effects, was announced recently by scientists of Parke, Davis & Company. The announcement, contained in the April 30 issue of Science, describes the new vitamin Bc as definitely a member of the Vitamin B complex.

The subletter "c" has been added to the name to represent the thousands of chicks on which experimental studies have been made.

Six Parke-Davis research men signed the paper announcing the isolation and crystallization of the new vitamin, fourteenth in the aristocratic vitamin family. They are Drs. Pfiffner, Binkley, Bloom, Brown, Bird, and Emmett.

Dr. J. J. Pfiffner has already attained prominence for his outstanding work on the suprarenal cortex. He prepared the first potent extracts for treatment of Addison's disease. Dr. A. D. Emmett is head of the company's division of vitamin and nutritional research and is a well-known vitamin authority. Dr. S. B. Binkley has hitherto been primarily associated with the isolation and synthesis of Vitamin K.

The existence of the antianemic Bc vitamin was first announced by Professor A. D. Hogan and his associates at the University of Missouri. They observed that chicks placed on a supposedly sufficient diet failed to grow and developed severe anemia, and that this could be cured by adding crude liver extract. Dr. Hogan and an associate, Dr. B. L. O'Dell, were able to make a 300-fold concentration of this antianemic factor from liver. Their work became the basis for extensive research which finally led to the isolation of this antianemic vitamin in pure form.

Parke-Davis research laboratories, headed by Dr. Oliver Kamm, scientific director, undertook the isolation of Vitamin Bc in the autumn of 1940. Their new, crystalline product represents a 500,000-fold concentration of the antianemic activity present in liver. Although the chemical formula of this newly-isolated vitamin is not known, obviously the next step on the Parke-Davis research program is its chemical identification. Studies are still being carried on, aimed at a larger production of Vitamin Bc so that eventually its value in treatment of various forms of secondary anemia can be determined.

Isolation of a vitamin is important because it offers hope of chemical identification and later the synthesis of the compound. It makes possible further advances in nutritional research since it can be incorporated into synthetic diets and thus serve as a tool for detecting the existence of additional vitamins.

For Milk-Allergic Patients

Appetizing and nutritious recipes for using Mull-Soy in milk-free diets are now available in a new publication of Borden's. Already widely prescribed as a hypoallergenic substitute for milk in infant formulas, Mull-Soy is now proving equally useful in diets of older infants, children and adults who are allergic to milk.

Mull-Soy is an ethically-marketed soybean food in liquid emulsified form. It is palatable, readily digestible, well-tolerated, and easy to use. Although hypoallergenic in most cases of milk allergy, it nevertheless closely resembles milk in nutritional values of protein, fat, carbohydrates, and minerals. Mull-Soy ingredients are entirely of non-animal origin, consisting of soybean, flour, soybean oil, soybean lecithin, dextrose, sucrose, calcium phosphate, calcium carbonate, salt, and water. After special processing at carefully controlled temperatures, the mixture is homogenized at high pressure, sealed in sanitary-type cans, and sterilized. In flavor it is slightly sweet and nutlike, and many find it makes a pleasing warm drink when simply diluted with an equal amount of hot water.

These Mull-Soy recipe folders are designed for distribution by physicians to their patients. Any desired number of copies may be obtained by writing to Borden's Prescription Products Division, Department CB, 350 Madison Avenue, New York 17, N. Y.

BOOK REVIEWS

A Manual of Clinical Therapeutics. By Windsor C. Cutting, M. D., Associate Professor of Therapeutics, Stanford University. Pp. 609. Cloth. Price, $4.00. Philadelphia: W. B. Saunders Company, 1943.

This Manual is almost entirely devoted to treatment, only a few lines being devoted to etiology or diagnosis. The discussions are short, as befits a Manual, but are ample. The therapeutics described is up-to-date and sufficiently detailed to be of ready use. The work includes tropical diseases as well as those current in this country.

The various appendices, covering over 150 pages, include such items as special procedures, physiotherapy, poisoning, diet lists, weights and measures, abbreviations, physiological data, dosages, etc.

The volume "is intended to be a comfort and practical guide for students, house officers, and practitioners of general medicine." It is such a guide, and a very superior one at that.

A Guide to Practical Nutrition. Edited by Michael G. Wohl, M. D., and John H. Willard, M. D. Pp. 98. Paper. Philadelphia: Philadelphia County Medical Society, 1943.

The editors, acting for their Society's Committee on Nutrition and Deficiency Diseases, have collected thirteen papers originally published in *Philadelphia Medicine* covering the major aspects of nutrition. These papers are entirely short, but are compact. In addition, in an appendix are charts of vitamins and food compositions. This little brochure is a valuable one to keep on the office desk, where it will answer immediately most of the questions your nutrition case presents.